Journal of Traumatic Stress, Vol. 17, No. 2, April 2004, pp. 163–171 (© 2004)

Twenty-Four-Hour Ambulatory Assessment of Heart Rate and Blood Pressure in Chronic PTSD and Non-PTSD Veterans

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This study examined 24-hr levels of ambulatory heart rate (HR) and blood pressure (BP) in 2 groups of male veterans: 19 with chronic posttraumatic stress disorder (PTSD) and 17 who never met criteria for PTSD. The relationships between diagnostic status, basal cardiovascular activity, and cardiovascular reactivity to stress were examined. Hierarchical linear modeling analyses revealed that the PTSD group had higher resting HR than the non-PTSD group. Moreover, the PTSD group showed greater BP reactivity during times of affective distress than the non-PTSD group. The health care implications of these findings are discussed, as are directions for future research.

KEY WORDS: PTSD; blood pressure; heart rate; ambulatory monitoring.

A relationship between posttraumatic stress disorder (PTSD) and basal cardiovascular activity has been observed (e.g., Blanchard, 1990). Specifically, in summarizing a series of studies which examined cardiovascular reactivity to trauma cues, Blanchard noted that PTSD groups consistently had higher baseline heart rate (HR) and blood pressure (BP) values than the non-PTSD groups. Given the increased risk for cardiovascular events (e.g., myocardial infarction) associated with elevated resting BP (Sherwood & Carels, 2000) and HR (Greenland et al., 1999), he surmised that PTSD may be a risk factor for cardiovascular disease. Consistent with these observations, a recent meta-analysis found that relative to comparison groups free of psychopathology, individuals with PTSD show elevated resting HR rate and BP in laboratory settings (Buckley & Kaloupek, 2001). Recent evidence also suggests that PTSD is associated with elevated rates of nonfatal myocardial infarction (Boscarino & Chang, 1999) and other indicators of cardiovascular risk (e.g., decreased heart rate variability; Cohen et al., 1997).

Several mechanisms have been proposed to explain the link between stress, basal cardiovascular activity, and ultimately, cardiovascular endpoints. For example, the cardiovascular reactivity hypothesis assumes that sympathetic activation associated with repeated and chronic stress promotes functional and/or structural changes in the cardiovascular system (e.g., damage to the endothelium which facilitates atherosclerotic buildup; Sloan, Shapiro, Bagiella, Myers, & Gorman, 1999). Indeed, studies show that relative to non-PTSD control groups, individuals with PTSD show greater cardiovascular responses to laboratory challenges that involve exposure to trauma-related cues (Blanchard, Hickling, Taylor, Loos, & Gerardi, 1994; Pitman, Orr, Forgue, De Jong, & Claiborn, 1987). Chronic PTSD also has been associated with abnormal noradrenergic function and hypothalamic–pituitary–adrenal (HPA) dysfunction such that individuals with PTSD are likely to have abnormally large sympathetic reactivity to stressful demands (McFall, Veith, & Murburg, 1992). Such marked

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cardiovascular reactivity to conditioned stimuli in laboratory settings has been associated with hypertension in non-PTSD samples (Frederickson & Matthews, 1990). This model, based on repeated and sustained stress reactivity, suggests that adverse changes in the cardiovascular system occur after long periods of time (Light, 2001) and predicts that only chronic PTSD will be associated with elevated HR and BP.

Another potentially fruitful explanatory model proposes that the association between PTSD and cardiovascular health is mediated by variables known to have direct effects on the cardiovascular system. For example, PTSD is associated with high rates of alcohol abuse/dependence comorbidity (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995). It is well documented that alcohol consumption of greater than three drinks per day is associated with increased BP and HR, and increased mortality from cardiovascular disease (Camargo & Rimm, 1996). These influences, and others like them (smoking), point to ways in which PTSD may have an indirect (mediated) relationship with cardiovascular health (readers interested in a complete review of PTSD and physical health are referred to Buckley, Green, & Schnurr, in press).

Notwithstanding the literature referenced above, one of the more compelling methods for investigating PTSD and cardiovascular health is underutilized. Specifically, ambulatory monitoring methods that measure cardiovascular activity in the natural environment have not been used much for examining the mechanisms that link PTSD to cardiovascular health. Such methods have been deemed more accurate predictors of cardiovascular risk than in-clinic assessments in cardiovascular-outcome research (Redon et al., 1998). Moreover, repeated measurement of cardiovascular indices allows for sophisticated examinations of person-variable (diagnostic status) and environmental-variable (stressful context) main effects as well as their interactions in the prediction of cardiovascular parameters (Schwartz & Stone, 1998).

Two studies have utilized ambulatory methods with PTSD samples and the findings are mixed. Muraoka, Carlson, and Chemtob (1998) found that relative to a non-PTSD comparison group, veterans with PTSD showed elevated basal HR, systolic blood pressure (SBP), and diastolic blood pressure (DBP). Beckham et al. (2000) utilized a similar methodology, but contrary to the Muraoka et al. findings, they noted no apparent differences in basal cardiovascular activity in veterans with and without PTSD.

Although helpful first steps, both Muraoka et al. (1998) and Beckham et al. (2000) studies examined aggregate measures of HR and BP (examined groups means collapsed across all assessments points). Several methodological critiques of this type of statistical analysis with repeated ecological measurements suggest that such analyses are not optimal for assessing person, environment, and person-by-environment interaction effects (Jaccard & Wan, 1993; Schwartz & Stone, 1998). Specifically, examining group means collapsed across repeated assessments violates statistical assumptions for several statistical tests and ignores unique variance that allows one to examine multiple influences on any given cardiovascular measurement (e.g., the interaction between personality traits and reactivity to stress at a given point in time). Accordingly, one cannot determine from previous work whether PTSD is associated with elevated HR and BP because of a true difference in basal cardiovascular activity, or as a function of greater frequency of environmental stress and potentiated cardiovascular reactivity to such stress. In addition, such approaches are less ideally suited to addressing the confounding metabolic effects such as group differences in activity and posture relative to hierarchical linear modeling methods (Schwartz, Warren, & Pickering, 1994).

Finally, no studies have examined the relationship between PTSD and magnitude of dipping of BP associated with sleep onset. Previous research demonstrates that a lack of such BP dipping (defined as a <10% drop in BP associated with sleep) represents a risk factor for cardiovascular outcomes (Ituarte, Kamarck, Thompson, & Bacanu, 1999).

In light of the aforementioned gaps in the existing literature, we collected 24-hr ambulatory cardiovascular measures in the natural environment of study participants. We examined two groups of veterans, those with chronic PTSD and those without PTSD. The groups were comparable on age, body-mass, gender, medication status, and family history of cardiovascular disease. We had the following specific objectives: (1) To provide a test of between-group differences in HR and BP in the natural environment of study participants while controlling for variables known to affect basal levels of cardiovascular parameters and those known to effect phasic changes in the cardiovascular system; (2) to examine the extent to which cardiovascular reactivity to environmental stress varied across diagnostic groups; and (3) to examine whether PTSD diagnostic status is associated with magnitude of dipping of BP at sleep onset.

Method

Participants

Participants were 36 Vietnam-era, male veterans and composed of 19 Vietnam combat veterans with current PTSD and 17 Vietnam-era veterans without PTSD. The

presence or absence of PTSD was determined by the CAPS interview (Blake et al., 1997; see below). The presence or absence of exposure to combat (the focal stressor for PTSD diagnosis) was verified by examination of the veteran's military discharge papers (DD214 documents). Combat during the Vietnam War was chosen as a selection criterion for trauma exposure in order to insure that our PTSD group met criteria for chronic PTSD (all experienced the disorder for >25 years). Medication status, substance abuse treatment history, and medical diagnoses were verified by review of each veteran's electronic medical record. Exclusion criteria for both groups were (a) diagnosis of diabetes mellitus; (b) current vasoactive medication use (beta-blocking agents, calcium channel blockers, and tricyclic antidepressants); (c) current psychosis; (d) current substance abuse or treatment for substance dependence within past 6 months; and e) history of myocardial infarction or stroke. Prospective participants were selected from a database of research volunteers maintained by the National Center for PTSD. The database contains relevant screening information such as medication status, age, years of education, PTSD diagnostic status, etc. Availability of screening information allowed a priori matching of likely PTSD-positive and PTSD-negative participants on key variables such as age and medication status. Structured interviews for establishing diagnosis were readministered on day 2 of the current study to insure diagnostic accuracy (see Procedure).

Participants were paid $250 for completing the entire protocol. Group demographics are presented in Table 1.

Table 1. Demographic and Covariate Scores for PTSD and Non-PTSD Subsamples

Variable	Group: PTSD (n = 19) M (SD)	Group: Non-PTSD (n = 17) M (SD)
Age	51.09 (3.33)	53.36 (3.10)
Education (years)	14.09 (2.30)	13.89 (2.27)
Body Mass Index	29.07 (4.44)	28.71 (4.66)
BDI score*	16.64 (9.83)	4.50 (3.70)
STAI-state score*	46.64 (9.85)	28.64 (8.46)
STAI-trait score*	49.82 (9.58)	28.64 (8.46)
MMPI2 Hostility T-score	61.73 (13.93)	54.93 (8.63)
AUDIT score	7.00 (8.77)	03.28 (5.65)
Ethnicity (% Caucasian)	64	86
Cigarette smokers (% yes)	36	21
Family history of CVD (% yes)	64	71

Note. BDI = Beck Depression Inventory, STAI = State/Trait Anxiety Inventory, MMPI2 = Minnesota Multiphasic Personality Inventory-2, AUDIT = Alcohol Use Disorders Identification Test, CVD = Cardiovascular disease.
*$p < .05$.

Assessment Instruments

The Clinician Administered PTSD Scale (CAPS; Blake et al., 1997) was used to determine PTSD diagnostic status. This semistructured interview assesses the 17 symptoms of PTSD defined by *DSM-IV* (American Psychiatric Association [APA], 1994), and is commonly used as a "gold standard" diagnostic instrument for assessing PTSD.

The MMPI-2 (Greene, 1991) was utilized to gather quantitative indices of various personality and behavioral traits. The Beck Depression Inventory-II (Beck, Steer, & Brown, 1996) was used to assess depressive symptomology. The State/Trait Anxiety Inventory (Speilberger, Gorsuch, & Lushene, 1970) was administered to assess both state and trait anxiety. The Alcohol Use Disorders Identification Test (AUDIT), a 10-item screening questionnaire developed by the World Health Organization (WHO) was used to provide a quantitative index of current alcohol use, with higher scores indicating problems with alcohol abuse (Saunders, Aasland, Babor, de la Fuente, & Grant, 1993). The Health Risk Appraisal (HRA; University of Michigan, 1989) is a 59-item self-report instrument that assesses family medical history and current health behavior practices such as smoking and dietary habits.

Cardiovascular Assessment and Equipment

In-Clinic Cardiovascular Assessment

A DinaMap automated blood pressure monitor (Model 1846SX) with an inflatable occlusion cuff was used to take in-clinic blood pressure measurements prior to the ambulatory monitoring phase of the study. Dinamap readings were used to calibrate the range of operation for the ambulatory blood pressure monitors.

Ambulatory Monitoring Equipment

DynaPulse 5000A (Pulse Metric) ambulatory monitors were used to collect HR, SBP, and DBP. These monitors utilize the oscillometric method of measurement with a transducer in the occlusion cuff. The occlusion cuff was placed on the nondominant arm at the level of the heart. The monitors were programmed to take measurements every 20 min during specified waking hours and every 120 min during sleeping hours. Patients wore the monitors for a full 24-hr period.

Activity Logs

During the ambulatory monitoring phase of the study, patients kept activity logs and recorded the following information on the day of ambulatory monitoring: caffeine intake, over-the-counter medication intake, cigarette smoking, and time of meals. Subjects also recorded posture, activity, stress ratings, and location for each specific HR and BP measurement. The log entries were utilized as time-varying covariates in subsequent statistical analyses.

Procedure

Upon initial contact with participants, staff conducted a short phone screen to determine eligibility for the study. Eligible participants were invited to take part in the study, which involved two visits to the clinic.

Day 1

Participants were provided with a description of the study and a consent form that had been approved by Institutional Review Board of the local VA Medical Center. Staff then answered any questions related to the project.

Participants' height and weight were measured using Detecto Physician's Scales. Staff then took five blood pressure measurements on the DinaMap monitor. The five clinic readings were taken with the subject in a seated position with both feet on the floor. The occlusion cuff was placed on the nondominant arm at the level of the heart. The measurements were taken at 1-min intervals.

Participants then spent 2 hr completing the questionnaires described above. No trauma-related material was discussed on day 1 to avoid introducing emotionally evocative events prior to conducting the ambulatory monitoring (which might artificially inflate the ambulatory readings due to elevated state anxiety). At the end of day 1, participants were instructed in the appropriate use of the ambulatory monitors and behavioral recording diaries. Participants were fitted with the monitors and three preliminary readings were taken with the ambulatory monitor before study assessment readings were initiated. The three preliminary readings were obtained and discarded with the aim of habituating participants to the monitor. This was done because previous research shows that the first few readings gathered during ambulatory monitoring phases can be artificially inflated before participants acclimate to the recording device (Owens, Atkins, & O'Brien, 1999).

Day 2

Participants returned 24 hr later to discontinue ambulatory monitoring. The data were then transferred to a desktop PC for analysis.

Assessment of PTSD was conducted on day 2. Participants completed the Combat Exposure Scale (Lund, Foy, Sipprelle, & Strachan, 1984) and the Life Events Checklist (Blake et al., 1997) to assess for the presence of both military and civilian trauma that might constitute a criterion A trauma as defined by *DSM-IV* (APA, 1994). After the trauma screen, the CAPS was conducted to determine the current and lifetime status of PTSD. All diagnostic interviews were completed by the first or second authors, who are doctoral level clinical psychologists. Subsequent to the day 2 assessment procedures, participants were debriefed and questions answered.

Data Analyses

Data Reduction and Artifact Detection

In accordance with published guidelines, the cardiovascular data were screened for artifacts (Marler, Jacob, Lehoczky, & Shapiro, 1988). The following algorithm was applied to the data to screen out artifacts: Systolic blood pressures outside the range of 70 to 250 mmHg were excluded; diastolic blood pressures outside the range of 45 to 150 mmHg were excluded. Any ratio of systolic/diastolic that was below $1.0625 + (0.00125 \times \text{diastolic})$ or above 3.0 was considered an artifact. Whenever an out of range value was found, the corresponding BP, HR, and diary values were removed for that measurement point.

Values for HR, SBP, and DBP were examined as a function of waking and sleeping hours. The cardiovascular data were tagged as either a waking measurement or sleeping measurement depending on the sleep interval reported in the behavioral diary.

Hierarchical Linear Modeling Analyses

In accordance with published guidelines for the analysis of ecological data with multiple measurements (Schwartz & Stone, 1998), we analyzed the data utilizing hierarchical linear modeling (Raudenbush & Bryk, 2002). Full maximum likelihood solutions were used for all analyses. This method allows for analysis of repeated

measures without requiring unduly strict assumptions about the equality of slopes and intercepts across the sample. The repeated measurements for HR, SBP, and DBP were the dependent variables for all statistical models. A separate model was run for each cardiovascular dependent measure (e.g., HR, SBP, DBP) for any specific study question addressed. Given that the amount of available data points varies for each subject with this type of methodology (due to differences in length of sleep cycle, number of missing data points, etc.), this data analytic approach is appropriate as it can accommodate an unequal number of data points per subject.

Behavioral diary data recorded for each ambulatory measurement (e.g., posture, stress rating) were used as time-varying covariates. Diagnostic group membership (coded dichotomously) and individual difference variables that were scaled on an interval level (e.g., Body Mass Index) were used as between person predictors of cardiovascular measures. In addition to main effects, interactions between the aforementioned variables were examined in accordance with the study hypotheses outlined in the Introduction.

HLM 5 Software was used to conduct the hierarchical linear model analyses (Raudenbush, Bryk, Cheong, & Congdon, 2001). Demographic variables and other descriptive statistics between the groups were summarized via independent groups t test or chi-square analyses in order to characterize our sample.

Selection of Predictor Variables

Our pool of time-varying covariates and person level predictors was kept small given the relatively small sample size. Specifically, in order to avoid inflating the predictor variable-to-subject ratio, we took an empirically informed approach to predictor variable selection. For example, because we age matched the two groups and restricted our sample to only Vietnam combat veterans, the range of our age variable is tight and well controlled (mean age of 52.8, $SD = 3.3$). Bivariate Pearson correlations indicated that age did not statistically covary with BP or HR in the manner one would expect if an unrestricted age range was utilized. Thus, age was not used as a covariate. Conversely, body mass showed positive statistical correlations with BP values and was used in the analyses.

Selection of predictor variables proceeded in this manner. In the end, body mass, smoking status, and minority status were the only person-level predictors that had statistically significant relationships to the outcome variables and thus, were used as person-level covariates. Posture was included as a time-varying covariate given the well-established effects of posture on HR and pressor levels.

Results

The demographic characteristics of the subsamples are presented in Table 1. On the basis of independent groups t tests or chi-square analyses for categorical data, the groups did not differ at a statistically significant level ($p < .05$) on age, education, percent minority status, body-mass, percentage of individuals who smoke tobacco, or percentage of individuals with a positive family history of cardiovascular disease. Not surprisingly, the PTSD group had uniformly higher means on measures of psychopathology.

Cardiovascular Measures

To test our first aim of the study, we conducted hierarchical linear modeling analyses whereby we examined the effect of diagnostic group membership (PTSD vs. non-PTSD) on waking cardiovascular measures after taking into account the effects of body mass, smoking status, and posture. To examine aim number two, we also examined the main effect of stress, as well as diagnostic group × stress interaction.

Between-Group Differences in Waking HR and BP

With respect to HR, PTSD diagnostic status accounted for a 6.63 beat-per-minute increase in heart rate beyond the effects of posture, body mass, smoking, ethnicity, and stress ratings. With respect to both SBP and DBP, there was no main effect of PTSD diagnostic group for either variable after controlling for individual difference covariates and time-varying covariates. A summary of these effects is presented in Table 2 with statistically significant effects marked by an asterisk. The intercept in these statistical models represents the grand mean before taking into account variables in the equation, whose direct effect can be determined by examining the size and direction of the unstandardized coefficient listed for that variable.

Cardiovascular-Reactivity to Stressful Demand

In order to examine the extent to which cardiovascular-reactivity to environmental stress varied across diagnostic groups we examined the interaction between

Table 2. Effects of PTSD, Stress, and Covariates on Cardiovascular Measures

	B	*SE*	*t* Ratio	*df*
Heart rate variables				
Intercept	69.62	2.26	30.73***	25
Diagnostic group	6.63	2.99	2.22*	25
BMI	−0.86	0.36	2.36*	25
Minority status	−4.93	3.71	−1.33	25
Smoking status	4.22	3.16	1.34	25
Stress	1.48	1.88	0.79	28
Group × stress interaction	−3.29	2.53	−1.30	28
Lying (vs. sitting)	−5.27	1.40	−3.76**	29
Standing (vs. sitting)	4.04	1.05	3.86**	29
SBP Variables				
Intercept	132.24	1.86	71.29***	25
Diagnostic group	1.35	3.83	0.35	25
BMI	1.27	0.51	2.52*	25
Minority status	20.33	7.45	2.73*	25
Smoking status	6.08	5.13	1.18	25
Stress	−2.99	2.76	−1.09	28
Group × stress interaction	6.54	3.12	2.10*	28
Lying (vs. sitting)	−9.18	1.72	−5.34***	29
Standing (vs. sitting)	1.29	1.40	0.92	29
DBP Variables				
Intercept	73.35	1.19	61.89***	25
Diagnostic group	0.08	2.19	0.04	25
BMI	1.12	0.31	3.66***	25
Minority status	10.81	4.42	2.45*	25
Smoking status	3.35	2.44	1.37	25
Stress	−4.53	2.55	−1.78	28
Group × stress interaction	9.71	3.21	3.02**	28
Lying (vs. sitting)	−6.31	1.28	−4.93***	29
Standing (vs. sitting)	1.35	0.82	1.65	29

Note. Coefficients are unstandardized. SBP = Systolic Blood Pressure, DBP = Diastolic Blood Pressure, BMI = Body Mass Index.
*$p < .05$. **$p < .01$. ***$p < .001$.

diagnostic group membership and affective distress (stress) for magnitude of responding in cardiovascular parameters. By adding the rating of whether subjects reported a stressor at the time of cardiovascular measurement, we were able to examine the main effect of stress and the diagnostic group × stress interaction. The results for these analyses can be found in Table 2. For the HR analysis this yielded a nonsignificant main effect for stress and a nonsignificant effect for a diagnostic group X stress interaction effect.

Although there was no main effect of stress on SBP levels, there was a significant diagnostic group × stress interaction effect, with the coefficient indicating a 6.54 mmHg difference in reactivity between the two groups (the PTSD group showed larger SBP reactivity when reporting stress than non-PTSD group). The effects for DBP also revealed no main effect for stress. There was however, a diagnostic group × stress interaction whereby the PTSD group showed a 9.71 mmHg greater increase in DBP while under stress than the non-PTSD group. Coefficients and test statistics are presented in Table 2.

PTSD and Dipping of Nocturnal Blood Pressure

With respect to reduction in blood pressure associated with sleep, we conducted an HLM analysis on blood pressure values from the entire 24-hr assessment period. A comparable analysis that was completed for the first two aims of the study was repeated with a sleep variable added to the equation. A dichotomous rating associated with each blood pressure value indicated if the reading was taken during sleep or waking hours. This variable was coded based on reported sleep intervals from behavioral diary data. This variable was also examined in an interaction term with diagnostic group to see if the effect of diagnostic group influenced the reduction of blood pressure associated with sleep onset. As expected, there was a main effect of sleep for both SBP, $t(34) = -3.03$, $p < .01$, and DBP, $t(34) = -4.04$, $p < .001$, reflecting a reduction in pressor levels. However, the diagnostic group × sleep interaction effects were not statistically significant, suggesting that the magnitude of reduction was comparable across groups.

Discussion

Results of this study indicate that when groups of veterans with and without chronic PTSD are compared on basal cardiovascular activity, PTSD appears to be related to elevated basal HR and greater BP reactivity to stressful demands. It is interesting to note that our data revealed group differences in basal HR that cannot be accounted for by the measured behavioral and metabolic influences on HR (body mass, smoking status, posture). The magnitude of the basal HR differences in this ecological study are similar to those found in laboratory settings (Buckley & Kaloupek, 2001). There does not appear to be a group difference in basal blood pressures when the same variables are taken into account, only differences in magnitude of BP response to reported stressful context. However, the null effects for basal blood pressure, as well as any null effect in this study, must be tempered by the fact that we had a relatively small sample size ($N = 36$) and had a fairly complex linear model. Thus, it may be the case that some main effect or interaction differences were missed by the study due to low power.

The sleep onset data suggest that chronic PTSD is not associated with a lack of BP dipping during sleep. In total, the data are consistent with the notions of reduced vagal tone in PTSD (Cohen et al., 1997) and heightened, sympathetically mediated, cardiovascular reactivity to stress in PTSD (Blanchard & Buckley, 1999).

In total these findings are consistent with previous qualitative and quantitative reviews of laboratory findings which find PTSD is associated with elevated basal heart rate relative to non-PTSD groups (Buckley & Kaloupek, 2001). However, unlike most laboratory studies, which suggest PTSD groups show potentiated cardiac responses to trauma cues, this study revealed that the magnitude of increases in HR rate in stressful contexts did not vary as a function of group status. However, it is important to note that we considered any stressful context (e.g., fight with spouse) as an occasion to measure cardiovascular responses stress rather than examine responses to trauma cues exclusively. In laboratory cue-reactivity studies there tends to be an exclusive focus on trauma cues. This is important as it has been demonstrated there is a considerable range of cardiovascular responding that can vary greatly as a function of the nature of the stressor as well as individual difference variables (Manuck, Kamarck, Kasprowicz, & Waldstein, 1995).

It is clear from our BP data that subjects with PTSD tend to show stronger vascular reactivity to stress than the non-PTSD group when assessed in the natural environment. Failure to achieve the same interaction of diagnostic status and stress on the HR data may have been due to the case that the cardiovascular reactivity pattern of our PTSD group is characterized by a stronger vascular than cardiac component. That is to say, the PTSD group may show greater changes in total peripheral resistance than the non-PTSD group (as opposed to large differences between the groups in cardiac output during stress as a function of increases in HR). Certainly there is a broad range of cardiovascular responding to stress that varies across individuals with some individuals being predisposed to more vascular than cardiac-based response patterns (Manuck et al., 1995). It may be that the chronic PTSD group is characterized by such a response style. Given previous findings of elevated norepinephrine levels in chronic PTSD samples relative to controls (McFall et al., 1992), this seems like a plausible hypothesis. Specifically, peripheral resistance is governed almost exclusively by the sympathetic tone and by norepinephrine in particular (Andreassi, 2000). A finding of elevated norepinephrine might also provide an explanatory mechanism for the elevated heart rate finding. Unfortunately, we did not estimate 24-hr levels of norepinephrine in this study.

Given that our groups were deliberately selected to be comparable on such variables as body-mass, smoking, age, and family history, our findings may not be generalizable to the magnitude of differences between chronic PTSD and non-PTSD in the general population. In particular, it is the case that many lifestyle habits known to be associated with cardiovascular health are indeed much more prevalent among individuals diagnosed with chronic PTSD. For example, smoking is more than twice as prevalent among individuals with PTSD relative to the general population (Beckham, 1999). Similar findings exist for alcohol abuse (Kessler et al., 1995). Thus, it is highly likely that both cardiovascular reactivity to stress and adverse lifestyle habits interact in a cumulative fashion to adversely affect the cardiovascular health of individuals with chronic PTSD (Schnurr & Jankowski, 1999). Thus, this study may have underestimated the overall magnitude of the effect of PTSD on cardiovascular parameters by constraining the true effects of adverse lifestyle habits that more frequently co-occur with the disorder.

The selection criteria we imposed may have influenced our results in another way. We excluded individuals with physician-diagnosed hypertension and those on antihypertensive medications. Thus, to the extent that PTSD elevates risk for hypertension, we may have restricted the range on the outcome variables by examining this phenomenon at the lower end of the distribution of effects. Again, this may have resulted in an underestimation of

true population effects, which might account for our lack of a main effect of diagnostic group on basal BP values.

A final limitation of the study was the manner in which we measured stress. Specifically, we asked for dichotomous ratings (yes or no) as to whether subjects felt "stressed" at the time of each cardiovascular measurement. We did not attempt to identify the nature of the stressor (e.g., fight with spouse vs. responding to trauma-related cues). Thus, we cannot infer from this study whether the effects we found are related to trauma-related physiological arousal, a general pattern of augmented responding to all stressors, or both. This is an issue that warrants further investigation in future studies.

It is apparent that elevations of pressor values (in response to stress) and basal HR comparable to those found in this study constitute a health risk in prospectively followed groups of individuals in the hypertensive range (as evidenced by increased probability of stroke and coronary heart disease; Collins et al., 1990; Greenland et al., 1999). These findings suggest that the effect of PTSD diagnostic status on basal cardiovascular activity in the hypertensive spectrum is worthy of future investigation.

The present findings are consistent with evidence that PTSD is adversely associated with cardiovascular outcomes (Boscarino & Chang, 1999) and a variety of other health problems (Schnurr, Spiro, & Paris, 2000). Findings such as these encourage behavioral health interventions for individuals with PTSD to mitigate detrimental effects to physical health associated with the disorder. Beyond conventional psychiatric therapy aimed at reduction of PTSD symptoms, lifestyle or wellness interventions groups might be used to help promote healthy lifestyle habits and provide secondary prevention of physical morbidity among individuals diagnosed with chronic PTSD. Moreover, exposure therapy and stress management therapy might help to reduce the cardiovascular reactivity component of this disorder, thereby dampening overall risk to cardiovascular health. Indeed, recent treatment outcome data shows that cardiovascular reactivity to trauma cues diminishes with successful psychosocial treatment of PTSD (Blanchard et al., 2002).

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